

PSYCHOLOGY TEACHERS UPDATE

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ADDICTION

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PSYCHOLOGY TEACHERS UPDATE

Psychology Teachers Update is designed to give a brief overview of the main developments in the different areas of psychology. There is a proliferation of journals and research, and it is very difficult to keep abreast of the latest trends, particularly in the many and varied areas of psychology.

Each issue of Psychology Teachers Update will cover a particular topic, and summarise the main research directions and findings in the last ten to fifteen years approximately. The aim is to give teachers the feel of what is happening in that area of psychology.

Psychology Teachers Update will appear three times a year in January, May, and September. Subscription costs £20 per year for three issues (or £7 each).

Forthcoming topics include practical applications; behavioural genetics; schizophrenia; and critical psychology and psychiatry.

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INTRODUCTION

Addiction is commonly used in relation to the compulsive use of psychoactive (often illegal) drugs. Psychoactive drugs are those that have a psychological effect, like heroin, cocaine, anti-depressants, and tranquillizers. Addiction can, however, be applied to other behaviours. The key is that addiction creates "a problem that is in need of resolution" (Toates 2004).

Different terms are used in relation to addiction. These include substance dependence, dependency syndrome, and substance abuse.

The term "addiction" is not used in the classification of mental disorders, but rather the term "dependence". In DSM-IV (APA 1994), dependence requires at least three of the following:

- Tolerance - need for more of substance, or less effect of same dosage;
- Withdrawal - physiological reaction to stopping the substance: eg heroin produces sweating, nausea, cramps, and convulsions;
- Taking larger amounts than intended;
- A desire to control intake;
- A large amount of time spent obtaining the substance;
- Giving up of normal activities for this;
- The continued use despite knowledge of the damage of use.

The concept of dependence is applied to substances only, not to behaviours in DSM-IV. Technically, DSM-IV includes behaviour like pathological gambling in the category of impulse-control disorders.

Physiological dependence exists with tolerance and withdrawal, and psychological dependence when these two are not present.

DSM-IV-TR (APA 2000) lists the following substances under Substance Use Disorders: alcohol, amphetamines, caffeine, cannabis, cocaine, hallucinogens, inhalants, nicotine, opioids, phencyclidine, sedatives/hypnotics or anxiolytics, and polysubstance/other/unknown. The distinction is also made between substance dependence, substance abuse (social problems from substance use), and substance intoxication (behavioural or psychological changes due to effect of substance). There are a total of 123 sub-categories of Substance Use Disorders in DSM-IV-TR (table 1).

<u>Substance</u>	<u>Number of sub-categories</u>
------------------	---------------------------------

Alcohol	15
Amphetamine	12
Caffeine	4
Cannabis	8
Cocaine	12
Hallucinogens	10
Inhalant	10
Nicotine	3
Opioid	11
Phencyclidine	9
Sedative/Hypnotics/ Anxiolytics	15
Polysubstance/Other/Unknown	14

Table 1 - Sub-categories of Substance Use Disorders in DSM-IV-TR.

One way to describe substance use is through a series of steps from use to regular use to misuse and harmful use, and finally, dependency. After misuse would the behaviour be classed as substance use disorder in DSM-IV (Iversen et al no date).

Some definitions of addiction include an excessive craving for the substance. But this can be problematic because some drugs produce no craving (eg LSD), and craving can occur outside addiction (eg food cravings in pregnancy) (Toates 2004).

Griffiths (1996) has widened the definition of addiction to include behaviours, like gambling. He also highlights "positive addiction" behaviours (eg hobbies) with benefits like escape from stress, pleasure and relaxation, and give meaning to life. Though whether positive behaviours can be addictions is open to debate.

Addictive drugs work on the biochemistry of the brain (focused around the synapse), and change the activity of parts of the brain. Psychoactive drugs work in three main ways:

- Mimic neurotransmitters;
- Stimulate release of neurotransmitters;
- Slow down or prevent the breakdown of neurotransmitters after the synapse.

A principal area of study in the brain is known as the "incentive pathway": mesolimbic dopaminergic pathway (deep in the mid and forebrain, and includes the amygdala) (Toates 2004). Addictive drugs increase

activity (usually dopamine) in this area. Table 2 summarises the basic neurochemical changes of key addictive drugs (Toates 2004).

<u>Drugs</u>	<u>Area of focus in brain</u>
Opiates eg heroin, morphine	Occupy receptors on dopaminergic neurons, and increase release of dopamine
Cocaine	Blocks reuptake of dopamine after synapse, thereby increasing levels of dopamine
Alcohol	Increase dopamine and natural opioid activity in brain
Caffeine	Increase dopamine through reduction of adenosine (adenosine normally suppresses neurotransmitters)
Nicotine	Increase dopamine, particularly in amygdala, and acetylcholine

Table 2 - Addictive drugs and effects on the brain.

It seems that different biochemistry and different parts of the brain are involved in the various aspects of drug-taking behaviour (Iversen et al no date) (table 3).

<u>Behaviour</u>	<u>Brain area/Biochemistry</u>
Drug-seeking behaviour	Dopamine in dorsal mesolimbic area
Pleasure/reward	Dopamine in ventral mesolimbic area; brain's opioids
Withdrawal	Noradrenaline

Table 3 - Different aspects of drug-taking behaviour.

In the case of caffeine, there is disagreement over whether regular coffee-drinkers are addicted. It is addiction in the sense of the need for it, and withdrawal symptoms when without it, but there is not the need to

gain ever-increasing doses to receive benefits which is typical of addiction (Thompson and Keene 2004).

PREVALENCE OF ADDICTION

The number of people who are addicted can be calculated in a number of ways:

a) Lifetime prevalence - Number who ever suffered at any stage during their life: eg 13% of US population for alcohol dependence (APA 1994);

b) Annual incidence rate - Number of new sufferers in last year: eg 1.8% in US for alcohol dependence and/or abuse (Anthony 2005);

c) Current prevalence - Numbers who are addicted at this point in time: eg 4% of US population and alcohol dependence (APA 1994).

The prevalence rates of addiction vary depending upon the study. Government figures on alcohol and drug dependence are often less than epidemiological studies. Some of the differences are due to the sample, and to definitions of the terms.

Government figures use self-reported questionnaires, and databases of known cases, like drug-related deaths. The Office for National Statistics keeps a database of drug poisoning deaths in England and Wales. Between 1993 and 2004, there were 16 088 deaths related to drug misuse (Morgan et al 2006). But these figures are not necessarily the same as addiction rates.

In terms of Government questionnaires, 14% of men and 9% of women in England and Wales in 2006 admitted to drinking alcohol every day (Lader and Goddard 2006). Such surveys are large scale with over two thousand respondents, but "drinking everyday" is again not necessarily the same as addiction.

A better measure, the British Crime Survey, estimated that there were four million users of illegal drugs in 2002-3 (Condon and Smith 2003).

Internationally, alcohol is the number one substance abused, accounting for 4% of the "global disease burden" (ie all diseases in the world) (WHO 2002). While the United Nations estimated that 185 million people worldwide had used an illicit substance in 2003 (Phillips and Lawton 2004).

In Finland, the lifetime prevalence of alcohol dependence was calculated as 7.9% of the population, and 3.9% for current prevalence (in last twelve months)

(Pirkola et al 2006). Current alcoholics were significantly more likely to be single, have higher education, and co-morbidity of depression and anxiety.

Methodological issues affect large-scale studies as shown by the fact that estimates of illegal drug use were up to five times higher in the National Survey on Drug Use and Health (NSDUH) compared to the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) in 2003 in the US (Grucza et al 2007). Though the two studies used similar sampling techniques.

Many studies of substance use are self-reported by interviewees, and there is no way of knowing if the truth is being told to the researchers. One study (Rockett et al 2006) cleverly got round this problem by using participants who were giving blood and urine samples (for other reasons) in hospital. Thus their self-reports could be compared to the actual amount of drugs in their system. Generally, individuals did not under-report alcohol use, but they did for other drugs (table 4).

	<u>MALE</u>		<u>FEMALE</u>	
	<u>Self-report</u>	<u>Screening</u>	<u>Self-report</u>	<u>Screening</u>
Any substance	60.9	69.1	43.9	55.7
Alcohol	45.9	46.6	25.1	25.5
Marijuana	10.6	21.4	5.9	13.0
Cocaine	1.1	6.5	0.6	3.5

(After Rockett et al 2006)

Table 4 - Examples of prevalence of current (30-day) drug use from self-reports and toxicological screening (% of respondents).

The problem with trying to measure the overall prevalence of addiction is that it is usually a hidden activity, often because of the illegal nature of some drugs. Estimates are many times based upon the numbers who are known about by treatment programmes and arrests. Anthony (2005) described the treated (and known) cases of addiction as the small visible part of the iceberg compared to the untreated (and unknown) cases.

A prime example is crack-cocaine use. Population surveys of marginalised groups are of limited relevance because users are often not included in the sample, and, even if they are, they may not admit to drug use. The

British Crime Survey, which is based on 14000 households, found that 0.2% of respondents were crack-cocaine users in England and Wales in 2001-2 (Aust et al 2002). This is approximately twice as many as figures based on those who present at specialist drug treatment services (Hope et al 2005).

Researchers have developed statistical methods for the indirect estimate of prevalence of behaviour among hidden populations. These include capture-recapture techniques (Hook and Regal 1995), and the ratio-estimation method. The former uses multiple sources of data that are known, and looks for the overlap between them. Then using statistical models, the unknown element is calculated. For example, the known sources for drug users could be arrests and drug treatment referrals.

The ratio-estimation method calculates the size of a hidden population from the known data using a particular formula. For example, if one in ten drug injectors use crack-cocaine, then the numbers using crack-cocaine can be calculated from knowing the size of the population of drug injectors. The statistics are more complex than this in practice.

Hope et al (2005) applied these two statistical methods to estimate the prevalence of crack-cocaine use in London in 2000-1 using three known data sources in the twelve London boroughs:

- Individuals at specialist drug treatment agencies (n = 2905);
- "Arrest referrals" (those assessed as having drug problems at time of arrest) (n = 1188);
- Attendees at A & E due to overdoses, and a survey of known injecting drug users (n = 531).

The known data gave an observed population of crack-cocaine users of 4117 individuals. After statistical modelling, the unobserved population was calculated at 16 855, giving an overall prevalence in London of 1.5% of the total population for crack-cocaine users. These figures suggested that only one in six crack-cocaine users presented for treatment, and one in ten were assessed at arrest. Despite the fact that the statistical modelling makes certain assumptions, the authors felt that they had "plausible estimates" of crack-cocaine use.

POLYSUBSTANCE USE

Many addicts are polysubstance users: ie using or addicted to more than one substance: eg alcohol and cocaine. It can also mean the combination of drugs together to enhance the effect: eg heroin and methadone, heroin and cocaine ("speedballs") (Iversen et al no date).

In one recent study, Degenhardt et al (2007) found that amphetamine use at 24 years old in Australia was triggered and maintained by polydrug use. In a ten-year study, it was found that 20% of amphetamine users over the previous five years were nicotine dependent, 20% alcohol dependent, and 30% cannabis dependent. Overall 12% of the sample had used amphetamines in the previous year.

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CAUSES OF ADDICTION

There are many different theories of addiction, both general and specific to one type of addiction like alcoholism. West (2001) attempted to categorise the many theories into five groups:

i) Theories that focus on conceptualisation and general processes - Theories here explain all forms of addiction within a theoretical framework: eg psychoanalytic theory (Hopper 1995), sociobiological explanation (Kofoed and MacMillan 1987);

ii) Theories that focus on effects of addictive stimuli - These theories concentrate on the factors related to certain stimuli that produce addiction: eg physiological changes producing pleasure as reinforcement of the drug-taking behaviour (Bozarth 1994);

iii) Theories that focus on individual susceptibility - Here the focus is upon why some individuals are more susceptible to addiction than others: eg genetic differences (Cunningham et al 1992) or early attachment problems (Jaeger et al 2000);

iv) Theories that focus on environmental factors - Theories concentrating on environmental and social factors that make addiction more likely: eg stress (Breslin et al 1995);

v) Theories that focus on recovery and relapse - These theories may combine ideas from the other categories, but their main focus is explaining why some individuals recover from addiction and others relapse: eg personality disorders (Verheul et al 1998).

The Gateway Theory (Kandel et al 1992) has been proposed to explain the progress from smoking to other drug use. It would be that early age tobacco smoking could render the individual pharmaceutically vulnerable to addiction to other substances (eg changes in dopamine levels make the individual susceptible to artificial pleasure-seeking), or individuals may progress through peer pressure or the willingness to experiment (Patton et al 2005).

In an Australian study between 1992 and 2003, Patton et al (2005) investigated the "reverse gateway": ie early age cannabis use encouraging progression to tobacco smoking. Just under 2000 14-15 year-olds (at the start of the study) were questioned at eight points during the study. Weekly cannabis use at the start of the study predicted tobacco smoking ten years later eight times more than non-cannabis use. While non-tobacco smoking but

cannabis users at 21 years old were three times more likely to tobacco smoke at 24 years old. Regular cannabis use also predicted alcohol dependence.

Biological Explanations

Phillips and Lawton (2004) placed addiction within the framework of a "natural" human desire for intoxication. The substances used may vary from culture to culture, and throughout history, but the motivation for intoxication is common between them. Non-human animals also seek intoxication. For example, during the Vietnam war, water buffalo were filmed grazing on opium poppies.

The motivation to intoxication may originate in the pursuit of pleasure, excitement, self-medication, or novelty.

GENETICS/BIOCHEMISTRY

There has been more research on the genetic basis to addiction for alcoholism. Family and adoption studies show a significantly greater rate of alcoholism among those with alcoholic parent(s) compared to controls (Nurnberger and Berrettini 1998). Specifically genes related to dopamine production have been highlighted.

A large scale US twin study estimated heritability of drug abuse as around one-third (Tsuang et al 1996), and as high as three-quarters from a small adoption study (Cadoret et al 1995). Other research has suggested that there are sub-types of addicts, and certain types are more likely to be inherited than others (eg type 2 alcoholism; Cloninger et al 1988).

Dopamine deficiency in drug addicts is also evident in pathological gamblers. Zack and Poulos (2007) gave twenty pathological gamblers and eighteen non-gamblers a drug (haloperidol - antipsychotic drug) that reduced dopamine. For the gamblers, it led to increased self-reported rewarding effects of gambling, greater desire to gamble later, and increased speed of perception of gambling words compared to a placebo (table 5).

Genes controlling serotonin seem to be involved in alcoholism, though the exact genes have not been identified (yet) (Zill et al 2007). This knowledge comes from two sources:

- Lower levels of serotonin-related substances in cerebrospinal fluid of alcoholics;
- Taking drugs that increase serotonin leads to a reduction in alcohol intake in both rats and humans.

	<u>Gamblers - drug</u>	<u>Gamblers - placebo</u>	<u>Non- gamblers -</u>	<u>Non- gamblers -</u>
Enjoyed 15-min slot machine game (0-10)	6.0	5.0	4.25	4.5
Post-game desire to gamble (0-10)	6.0	4.5	1.0	1.0
Time to recognise words (millisecs)				
Gambling	895	950	998	1060

(After Zack and Poulos 2007)

Table 5 - Summary of results from Zack and Poulos (2007).

But in a British twin study, Fowler et al (2007) proposed that the common environment tended to be important in the initiation of cigarette, alcohol and marijuana use, while genetic influences were relevant for heavy use.

PHYSIOLOGICAL DIFFERENCES OF ADDICTS

Before Addiction

One area of explanation for addiction being explored is the idea that addicts are physiologically different in some way to non-addicts, and this difference(s) is the basis of the motivation to addiction.

This idea has been applied, in particular, to understand gambling addiction. A key difference between gamblers and non-gamblers could be related to level of arousal. For example, Brown (1986) proposed that gambling produces an optimum level of arousal. The pleasure of this optimum arousal acts as a reinforcer for the gambling behaviour.

Moodie and Finnigan (2005) focused upon measuring heart rate among three types of fruit machine gamblers in Glasgow city centre. The researchers recruited twenty-one each of frequent, infrequent and non-gamblers. Allocation to groups was based on the score from the South Oaks Gambling Screen (SOGS) questionnaire (Lesieur and Blum 1987). A score of five or above is classed as

pathological gambling. The frequent gamblers had a mean of 9.38, infrequent gamblers 2.52, and 0.19 for non-gamblers.

Autonomic arousal was measured through a "Finger Clip Probe" which records changes in blood pressure.

The frequent gamblers showed a significant increase in arousal from the baseline during and after playing on the machines, while non-gamblers showed a decline over the same period. Only the frequent gamblers continued to show increased arousal after playing (table 6).

	ALL GAMBLERS	FREQUENT GAMBLERS	INFREQUENT GAMBLERS	NON-GAMBLERS
Before play	77.25	75.10	75.50	81.1
During play	79.90	80.60	77.87	81.3
After play	78.25	83.2	75.43	76.10

(After Moodie and Finnigan 2005)

Table 6 - Mean heart rate of fruit machine gamblers.

Frequent gamblers also showed other arousal differences: greater increase in arousal for wins, bonus plays, nudges, and features (eg backspace). These characteristics of the machines give the illusion that skilled is needed (Griffiths 1994). Frequent gamblers won only slightly more than the other two groups in this study.

After Addiction

An alternative possibility is that the drugs taken produce physiological differences in the addicts. In Robinson and Berridge's (1993) Incentive-Sensitization Theory, the drug changes the brain and produces a high motivation towards the objects associated with the drug (eg syringes). The brain is actually more sensitive to these cues because more neuronal connections have developed in relation to them (ie a strengthening of excitatory synapses on dopaminergic neurons).

In the language of the theory, stimuli associated with drug-taking are attributed "incentive salience": ie these stimuli become wanted even if the expectation of drug pleasure is reduced after repeated use. It is almost as if the craving is for the associated aspects of the drug - the means of taking (eg syringes) and the environment (eg "shooting galleries").

For Robinson and Berridge, craving is key to

addiction because it persists "long after discontinuation of drug use", and "as craving for drugs increases the pleasure derived from drugs often decreases" (p249).

The sensitization process varies between individuals due to genetic, hormonal or experiential factors (Robinson and Berridge 2001).

Based on neuroimaging studies, Volkow et al (2004) suggested that four brain areas are involved in addiction - the areas related to memory, motivation, reward, and control. Exposure to the drug or drug-related stimuli triggers the memory of rewards, and this stimulates the motivation and reward areas, and reduces the activity of the cognitive control regions of the brain.

Other Differences

Another difference could be between the adult and adolescent brain. Work with rats that injected tetrahydrocannabinol (THC) (the active ingredient of cannabis) over eighteen days found that adolescents preferred it to adults. However, the adolescents' hippocampus was more affected leading to short-term memory problems (Quinn et al 2007 quoted in In Brief 2007).

Miller (2001) has proposed a different explanation for addiction to many others under the heading of toxicant-induced loss of tolerance (TILT). This draws a similar origin for addiction and abdiction (multiple chemical intolerance eg "Gulf War Syndrome"). Prior to TILT, an individual responds normally to a substance with limited stimulant or withdrawal effects. Following TILT (ie after a major chemical exposure), responses are amplified, and to avoid unpleasant withdrawal symptoms the individual avoids the substance (abdiction) or consumes it regularly (addiction). Put another way, the desire to avoid withdrawal is the motivation to addiction, but only after the body has changed due to a major chemical exposure.

BRAIN DIFFERENCES

Neuroimaging studies are able to show the effects of drug abuse upon the brain (structural and chemical), but also the effects prenatally. In other words, the brain changes due to pregnant mothers abusing drugs. For example, children with prenatal methamphetamine exposure showed smaller areas in the basal ganglia (or striata) than the age norms (Chang et al 2007).

Using structured magnetic resonance imaging scans,

Chang et al (2004) compared thirteen children exposed prenatally to methamphetamine and fifteen unexposed children at age seven. The exposure to methamphetamine was for at least two-thirds of the pregnancy. The exposed group had an average 20% smaller hippocampus volume. The researchers were not sure the long term implications for the children of these differences in brain structure.

Dom et al (2005) reviewed a number of neuroimaging studies on the orbitofrontal cortex of addicts. The researchers found clear differences in function and structure of addicts compared to non-addicts, particularly manifest in deficits in decision-making. Are these differences a consequence of the addiction or a cause of it? Dom et al concluded that "the findings reported on in this review seem to be relatively independent of the type of substance misused. The observed abnormalities are probably not substance-specific but rather constitute a common deficit in addicted states and/or a common predisposing vulnerability.." (p218). Similar abnormalities in the orbitofrontal cortex have been reported in gambling addicts (eg Cavedini et al 2002).

The decision-making deficit is tested in the lab by card games to "earn" money, and require the ability to learn associations between reward and punishment among the cards. Failure to learn these associations could explain individuals starting drug use without weighing up the benefits and consequences fully.

Scans using functional magnetic resonance imaging reported a variety of brain differences among cocaine and amphetamine (stimulants) dependent individuals (Aron and Paulus 2007).

In terms of reward learning in drug addiction, increased dopamine is involved. For example, cocaine binds to molecules called dopamine transporters, and prevents removal of dopamine after the synapse. Dopamine is usually involved in changing neurons to remember rewards, and increased dopamine reinforces strongly the reward of drugs like cocaine. Normally once a behaviour is learned, dopamine is not released unless something new happens. But cocaine means that dopamine continues to be released every time the drug-taking behaviour occurs and amplifies its effect:

In this way, cocaine addicts "over-learn" behaviours associated with the approach and acquisition of cocaine. This over-learned behaviour, both drug-seeking itself and the intrinsic thinking associated with drug-seeking (eg craving), is not easily disrupted by other stimuli and ultimately successfully competes with biological rewards (Kalivas 2007 p72).

A consequence of long-term use of drugs like cocaine

is that the parts of the brain that use dopamine become less flexible. In other words, patterns of behaviour become fixed and rigid, particularly related to the drug-taking behaviour.

PERSONALITY DIFFERENCES

The idea of an "addictive personality" is attractive. Though most researchers feel this is too simple, there are individual differences between addicts and non-addicts on personality traits like impulsivity and sensation-seeking.

Differences between addicts of different substances exist according to some studies. For instance, alcoholics are more impulsive than non-alcoholics, but less impulsive than cocaine and heroin addicts. In turn, heroin addicts are more impulsive than cocaine addicts. Other studies find no differences between them all (Conway et al 2003).

It is easier to look at polysubstance abusers than individual substance abusers for personality differences compared to non-abusers. Conway et al (2003) concentrated upon 325 individuals in Connecticut, USA receiving treatment for heroin, cocaine and/or alcohol addiction.

Four measures of personality were taken for each participant using:

- a) Temperament and Character Inventory (TCI) (Cloninger et al 1994) - 226 true or false items measuring characteristics like novelty-seeking;
- b) NEO Five-Factor Inventory (Costa and McCrae 1992) - 60 items on a five-point scale;
- c) MacAndrew Alcoholism Scale (MAC) (MacAndrew 1981) - 51 items measuring things like impulsivity and disinhibition;
- d) California Personality Inventory socialization scale (CPI - soc) (Gough 1987) - measures of social adherence, for example, on 46 items.

Polysubstance abusers were found to be different depending on the number of substance abused (two or three) compared to single substance abusers. Polysubstance abusers had higher novelty-seeking scores on TCI, greater tendency towards social deviance on CPI-soc, and more impulsivity on MAC, but no differences on Neo Inventory. Conway et al felt that the common characteristic was behavioural disinhibition for polysubstance abusers (and this may have a biological basis).

Childhood and Social Factors

A number of childhood risk factors have been found in the trigger to drink alcohol, and the progression to dependency. Age of first drink is key: 15.7% of those who drank alcohol before age 15 were rated as alcohol dependent at 21 years old compared to 7.2% of post-15 first drinkers (Dawson 2000). Early drinking also goes hand in hand with adolescent problem behaviour, like conduct disorders. While alcoholism in the parents has different effects: maternal alcoholism influences the transition from occasional to regular drinking, and parental alcoholism the progression from regular to problem drinking (Lieb et al 2002).

Sartor et al (2006) found that, apart from conduct disorders, different factors were involved in triggering the first drink, and in the transition to alcohol dependence. Sartor et al used over 1200 male twins from the Vietnam Era Twin Registry in the USA. Early first drink of alcohol was associated with ADHD, both parents alcoholics, and parental divorce. Transition to alcohol dependence was associated with nicotine dependence, cannabis use, and generalised anxiety disorder. Conduct disorders were associated with both first drink and alcohol dependence.

Focusing on early childhood experiences as key, Flores (2004) conceptualised addiction as an attachment disorder. The inability to form attachment (from childhood) to help regulate emotions leads to individuals using substances to regulate emotions. The substance abused reduces further the possibility of forming attachments, and thus a cycle of addiction is set.

ROLE OF MEDIA

By the end of high school in the US, 80% of students have tried alcohol and 50% are current drinkers (Johnston et al 2002 quoted in Ellickson et al 2005). There is a concern that advertising of alcohol both directly on television and in magazines, and indirectly through sports events encourage adolescent drinking. Establishing whether alcohol advertising encourages drinking is difficult, and often retrospective studies are used. This type of study asks participants to recall advertisements from when younger, and correlates this with current drinking behaviour. Prospective studies are better. This method follows a group of participants for a period of time, and attempts to predict future behaviour from the current situation. One such study is Ellickson et al

(2005).

Based on forty-one middle schools in South Dakota, USA, 3111 seventh-graders (12/13 years old) were followed for two and a half years. It was part of ALERT drug prevention programme at the schools. Four types of alcohol advertising were studied - television, in-store displays, magazines, and concession stands at sports and music events.

At baseline, the students were divided into those who had never drunk alcohol (n = 1206) and those who had tried alcohol (n = 1905). At the end of the study, alcohol consumption in the last year was measured (0, 1-2, 3-10, 11-20, more than 20 times). An exposure index was calculated for the amount of alcohol advertising seen based on, for example, television viewing, and attendance at sports events.

The study found that alcohol advertising exposure did predict later adolescent drinking, but the type of advertising varied between drinkers and non-drinkers at grade 7 (table 7). Overall television advertising of alcohol had the least effect compared to other types of advertising.

	<u>Non-drinkers at grade 7</u>	<u>Drinkers at grade 7</u>
Reported using alcohol in past year at grade 9 (%)	48	77
Exposure to alcohol advertising		Greater for all four types
Type of alcohol advertising having greatest effect	In-store displays eg supermarkets	Magazines, concession stands
Role of non-advertising factors	Both - adult approval of drinking Only - weak bonds with school	Both - insufficient parental monitoring Only - peers who drink

Table 7 - Alcohol advertising and adolescent drinking.

The researchers were hoping to develop alcohol prevention programmes to counter advertising, but admitted how difficult it was:

Most adolescents go to the supermarket or corner food store on their own or with their parents or other adults; in many states, they also see alcohol linked with the necessities of life. Others see alcohol promotion in liquor stores, when they participate in "good time" outings such as sports events and music concerts, and when they read sports, news, music and other magazines (Ellickson et al 2005 pp244-5).

The role of the media in encouraging alcohol use does not have to be direct in the form of advertising, but it can be indirect through music. Herd (2005) performed a content analysis on 341 rap music song lyrics from 1979 to 1997. Over the period of the study, there were increasing references to alcohol (8% to 44% of songs), positive attitudes towards alcohol (43% to 73%), and brand names mentioned (46% to 71%). Overall, alcohol use was presented as having positive consequences. For example, 40% of the more recent songs (1994-7) presented positive consequences like relaxation, increased social status, and social acceptance for alcohol use compared to 13% of songs including negative consequences like health problems (and none mentioned addiction).

Herd concluded that it was "possible that using rap artists to advertise alcoholic beverages has helped to introduce and normalise alcohol use as part of the lifestyles portrayed in rap music" (p1266).

SOCIAL CONTEXT OF ADDICTION

There is a social context to drug-taking, in terms of the norms and discourses related to it, particularly for socially acceptable substances like alcohol. The discourses around alcohol are contradictory in today's society - "have a great time" versus "health risks".

In a qualitative study, de Visser and Smith (2007) found evidence of this ambivalence towards alcohol among thirty-one young London men. The motives for not drinking (violence, alcoholism, and cost) expressed by the men were also motives for drinking: "for example, positively evaluated confidence accompanying mild intoxication could easily become negatively evaluated arrogance following excessive consumption" (p354).

One interviewee, "Daniel" encapsulates the ambivalence of being drunk. On the positive side: "You never know where you're gonna end up. That's what I like about it". But on the negative side: "Like I wake up in the morning, roll over and I see this, like, ugly fucking dog looking at me.." (p355).

The existence of contradictory ideas in relation to

alcohol shows that its consumption is a narrow line to be walked. Positive discourses encourage consumption, in some cases in large amounts, for fun and escapism, but it is important never to go too far. How far is "too far" is a socially constructed concept, and varies between individuals and social groups, but it also shows the socially constructed nature of addiction, certainly for socially acceptable drugs.

A social constructionist position would challenge the concept of addiction as something definite that exists or does not exist. Addiction is a fluid concept which is constructed in many and varied ways in society.

CONCLUSIONS ON CAUSES

Choice and Determinism

The degree of choice that individuals have towards taking the substance is hotly debated. A biologically deterministic view, like Blum (1991), argued that a physiological deficiency predisposes the individual to addiction. In this case, alcohol is taken to negate the neurochemical imbalance in the brain's opioid system.

While Cox and Klinger (1988) are less deterministic. They see individuals as deciding to drink alcohol or not "on the basis of whether the positive affective consequences that they expect to derive from drinking outweigh those that they expect to derive from not drinking" (p170). This view fits with "rational models" of addiction, which even see addiction as a myth at the extreme (Davies 1992).

Recently, West (2006) attempted to bridge the determinism debate with the synthetic theory of addiction, which combines learning, compulsions and desires, self-control, beliefs, decisions, and plans.

Combination of Ideas

Generally, recent models of the cause of addiction have moved away from single explanations to a combination of factors. These can be divided into:

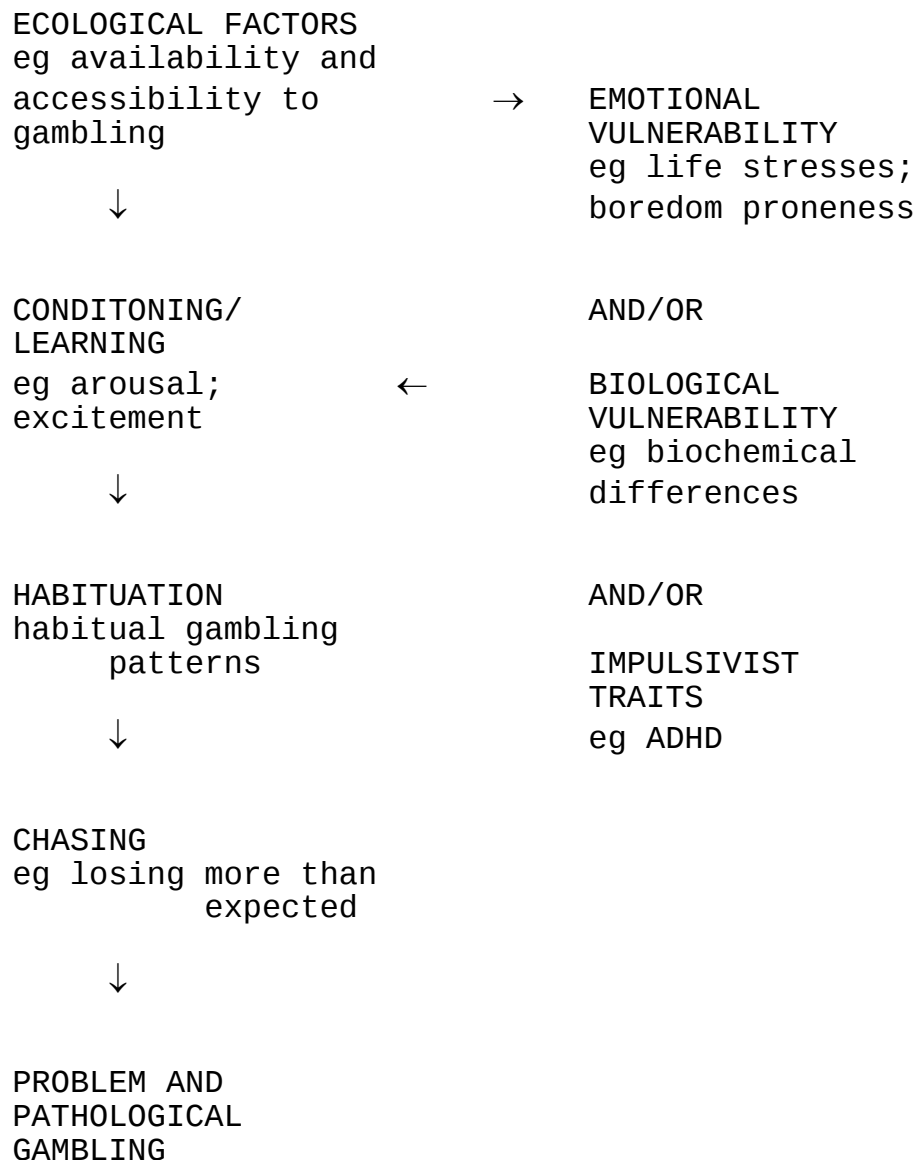
- i) Antecedents or precipitating factors: eg family history of addiction, lack of attachment in early childhood;
- ii) Triggers for starting drug use: eg peer substance use, risk-taking or rebellious adolescent behaviour;
- iii) Maintenance of dependence: eg poor social opportunities, positive attitudes towards drugs.

Chassin et al (1999) developed statistical path coefficients to predict alcohol and drug problems among 246 adolescents with at least one alcoholic biological parent and 208 controls after a five to seven-year follow-up.

For the development of alcohol problems, adolescent alcohol use, paternal alcoholism, and adolescent external symptoms were significant predictors. Adolescent external symptoms were manifestations of problems as anti-social behaviour, for example, and this was predicted from parental alcoholism and anti-social behaviour. But those with adolescent internal symptoms (eg depression) did not predict alcoholism. A similar pattern was found for the development of drug use.

So how the child/adolescent deals with growing up with alcoholic parent(s) is important. If they internalise the experiences in the form of mental problems, alcoholism or drug use tends not to develop, but if they externalise the experiences as problem behaviour, alcoholism and drug problems are more likely.

Blaszczynski and Nower (2002) combined the different theories on gambling into a pathway model. The model combines social (ecological) factors, conditioning, and vulnerabilities to explain the progress to problem and pathological gambling (figure 1).



(After Blaszczynski and Nower 2002)

Figure 1 - Pathway model of problem and pathological gambling.

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TREATING ADDICTION

PHARMACOLOGICAL TREATMENT

One issue with treatment, particularly of alcoholism, is whether "improved drinking" can last. In other words, whether abstinence is best after treatment. Mann et al (2005) followed-up patients sixteen years after alcohol dependence treatment at a German hospital. Abstinence from alcohol produced less relapse into alcoholism than reduced drinking after treatment.

O'Brien and McKay (2002) defined successful treatment as "the ability to function according to one's societal role".

But the best outcome for treating addiction to substances other than alcohol is abstinence. This is not easy with powerful drugs, and has led to the use of agonist maintenance treatment (ie: substitutes like methadone for heroine addiction) (table 8).

Methadone
levo-acethylmethadol
codeine
slow-release morphine
diamorphine
buprenorphine

Table 8 - Drugs used in agonist maintenance treatment.

Basically these substitutes maintain the biochemical changes in the brain produced by the addictive drug. This type of treatment prevents withdrawal, reduces craving, limits illegal drug use and criminal activities to gain money, and improves the addict's health (Haasen and van den Brink 2006).

There are these benefits to replacing one drug with another, but also there are problems, like side effects. For example, a small number of cases were found of Torsade de Pointes (fatal heart problem) with using levo-acethylmethadol with heroin addicts (Haasen and van den Brink 2006). Other problems include patient nonadherence, and abuse of the substitute (eg buprenorphine).

Methadone is the most effective substitute for heroin addicts, particularly while individuals are waiting to join a comprehensive treatment programme (Schwartz et al 2006). This can be especially important in prisons, for example. Dolan et al (2005) found a reduction in mortality, re-incarceration rates, and hepatitis C infection with such programmes in a study of Australian male prisoners.

The issues are around the level of daily dosage of methadone, and whether psychosocial care is needed as well (Haasen and van den Brink 2006).

Some treatment programmes prescribe substitutes with controlled amounts of heroin. This does seem to reduce criminal behaviour, but is not always acceptable to the general public, even if the addicts' health risks are also reduced (Haasen and van den Brink 2006).

O'Brien and McKay (2002), after reviewing the randomised controlled trials, concluded that nicotine, alcohol and opiates respond to pharmacological treatment, but not necessarily cocaine dependency.

OTHER TREATMENTS

Cognitive-behavioural therapy (CBT) focuses upon the thinking processes (particularly irrational beliefs) related to addiction. The aim is to change these by, for example, making the risks clear. In relation to gambling, say, participants would be given warning messages about irrational gambling beliefs. This has increased knowledge, but not necessarily changed behaviour (eg Steenbergh et al 2004). One exception is Floyd et al (2006).

They designed a lab experiment around a computerised roulette game with 120 undergraduates at the University of Memphis. The experimental group viewed a video about gamblers' irrational beliefs, and frequent warnings appeared on the computer screen during the game (eg "The result of any spin has nothing to do with previous spins"). The control group saw a video about the history of roulette and had no warnings. The experimental group demonstrated less risky gambling behaviour than the control.

But this was a lab study, and it did not include problem gamblers. The average number of times gambled in the past year was eight for the participants.

Treatment, not surprisingly, focuses upon the addict. But this is a consequence of addiction upon the family. Halford et al (2001) looked at ways to reduce stress in female partners of male problem drinkers in an Australian study. Sixty-one married women were randomised to one of three conditions: (i) supportive counselling; (ii) stress management; or (iii) alcohol-focused couples therapy. Each condition had fifteen one-hour sessions, and various measures were taken pre-test, immediately post-test, and six months after. All three conditions reduced the stress for the women, but there was no reduction in male alcohol consumption or improvements in "relationship functioning".

RECOVERY AND RELAPSE

Treatment for addiction is often received by only a minority: eg 25% of alcoholics in the USA (Dawson et al 2005). Whether individuals receive treatment or not, in terms of alcoholism, a number do recover, and this is aided by factors like being married, being female, and having non-alcoholic social support (Dawson et al 2005).

Using data from the 2001-2 National Epidemiologic Survey on Alcoholism and Related Conditions (NESARC) in the US, and DSM-IV definitions, Dawson et al (2005) calculated that 75% of alcoholics had shown some improvement over the past twelve months. Of the total sample, 27.3% had partial remission, 11.8% showed "asymptomatic risk drinking" (behaviour that produced risk of relapse, like intermittent heavy drinking), 17.7% low-risk drinking, and 18.2% abstainers. It was the last group who reported having received treatment most. The still alcohol dependent and abstainers were most likely to have dependency on other drugs.

Because of the limited number of people receiving treatment, it means that many people "drift" or "mature out" of drinking problems over time. However, the figures may be distorted because chronic or severe alcoholics are more likely to die than recover.

A lot of interest has centred on relapse prevention. Marlatt and Gordon (1980) noted that 23% of alcoholic relapses involved social pressure, but 29% were due to frustration and the inability to express anger. This has led to the use of social skills training to prevent relapse. For example, in a Norwegian study, hospitalised alcoholics given social skills training, as well as the usual treatment for alcohol, drank two-thirds less than the control given treatment for alcoholism only over one year. They also had 97% of work days sober compared to 45% for controls (Eriksen et al 1986).

Other treatments used with alcohols include communication training, behavioural self-control, and coping skills as preventive measures (Emmelkamp 1994).

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BEHAVIOUR ADDICTION

GAMBLING

Pathological gambling is characterised by an inability to control gambling which leads to personal, family, financial, occupational, and legal consequences (Blaszczynski and Nower 2002).

DeFuentes-Merillas et al (2003) investigated adult scratchcard buying in Holland. Of the sample interviewed, 28.4% were regular gamblers, 2.68% potentially problematic, and 0.24% had definable addiction. In the latter group, young women (25-34 years) with low income were the dominant group addicted to scratchcards only.

Blaszczynski and Nower (2002) identified three sub-groups of pathological gamblers - behaviour conditioned, emotionally vulnerable, and anti-social impulsivist.

The cognitive processes of gamblers can be different to non-gamblers, but there are also common processes like reacting to positive outcomes differently to negative ones. For example, individuals respond differently to being told that there is a one in fourteen million chance of being killed next Saturday evening (negative outcome) as opposed to the same odds of winning the National Lottery on the same day (positive outcome). This is the tendency to overestimate the likelihood of positive outcomes (Griffiths 1997a).

Tversky and Kahneman (1981) offered their participants the choice of a sure gain of £250 or a 25% chance of £1000. The majority (84%) went for the certainty of the former option. But when faced with choice of a sure loss of £750 or a 75% chance of a £1000 loss, the majority (87%) took the latter option. For the researchers, "losses loom larger than gains", and this can explain why gamblers take riskier bets after a series of losses ("chasing losses"). For example, after losing £10 each time on 14 races, the gambler is more likely to take odds of 15:1 on a £10 bet in the last race to break even compared to taking the same odds in the first race.

Another cognitive process is the tendency to attribute personal skill to successes despite the fact that gambling is entirely random. Choosing four of the six numbers correct in the National Lottery (ie nearly winning) is perceived as the result of skill, and improved skill will lead to winning next time.

In a classic participant observation study, Henslin (1967) mixed with dice players ("craps shooting") in the US. Henslin learnt how players attributed skill to their throw - a hard throw produces a large number and a gentle

throw a low one.

Similarly, individuals given random numbers are less confident of success than when they choose the numbers in a lottery game (Langer 1983).

FRUIT MACHINE GAMBLING

Fruit machines use the partial reinforcement effect (Wanner 1982) whereby rewards are intermittent but unpredictably regular. The feeling is that this time could be the next win.

Griffiths (1991) made the first extensive observation of adolescent fruit machine gambling in thirty-three UK amusement arcades (in seven towns: four inland and three seaside). Over 28 months, each arcade was visited between two to five times and observed for between 30 minutes and three hours, either during the day (11am-5pm) or in the evening (6-9.30pm). A total of 874 individuals were observed covertly during eighty observation sessions, of which over three-quarters were male players. The players had minimal social interaction (with little talking except to the machine) while playing, and regular players were very fast (eg 100 times in 10 minutes).

When a group played the machine, Griffiths observed a hierarchy with the most "skilful" players given status. It was not acceptable for subordinates to offer advice to such players.

Griffiths divided the motivations for playing fruit machines into:

- "Choice limitation" - "nowhere else to go";
- "Social factors"/"atmosphere" - meeting friends as "everyone goes down there";
- "Fun" - to "get a buzz";
- "Control" - a feeling of mastery over the machines and achievement in winning;
- "Escapism" - to "forget about home";
- "Winning money" - including cheating.

There were differences in clientele between inland and coastal amusement arcades.

By Griffiths (1996), two types of addiction to fruit machines had been formalised:

- Those who play to test skill, gain social rewards or "high";
- Playing for escapism because depressed and socially isolated. Machines seen as "electronic friends".

Dowling et al (2005) distinguished three types of gaming machines:

- i) Pachinko machines - played mostly in Japan, and accounting for nearly 60% of the world gaming machine market. It is a form of pinball where winnings are exchanged for non-cash prizes;
- ii) Amusement machines (eg fruit machines) - most popular in Europe and account for about 30% of the world market;
- iii) High-intensity gaming machines (eg video poker) - dominant in North America, and involving greater spending and speed of play than the other two types.

The initial research on gaming machines assumed "that all forms of gambling are equivalent and that findings related to one form of gambling can be generalised to other forms" (Dowling et al 2005 pp35-36). This is not seen as the case today.

One difference is the highly addictive nature of gaming machines (called the "crack-cocaine" of gambling in the media). A number of reasons have been proposed for this:

- a) The speed of development of pathological gambling

Breen and Zimmerman (2002) calculated that the progression to pathological gambling took about one year for machines, but three and a half years for other forms of gambling. This is probably true for a sub-set of gamblers with inherent vulnerabilities than the norm (Dowling et al 2005).

- b) Higher prevalence of gambling problems among machine users

For example, a national study in Australia (Productivity Commission 1999 quoted in Dowling et al 2005) reported gambling problems among 23% of regular machine gamblers and casino table players, but only 15% of racing gamblers and less than 6% of regular scratchcard players. In the same study, the amount spent on type of gambling produced gaming machines as the most prominent with problem gamblers.

Other researchers are not convinced that gaming machines are the most addictive. Griffiths (1997b) has argued that scratchcards are a "hard form" of gambling, though they appear innocent. This is because:

- There is a "short pay-out interval" (ie immediate for small amounts);
- "Near misses" act as an "immediate reinforcer" of the behaviour;
- Suspension of judgement: eg small stakes give the impression that less lost.

c) Gaming machines attract problem gamblers

The ease of access encourages problem gamblers to play these machines. However, gaming machines are far more popular with non-problem gamblers (Dowling et al 2005).

d) More gaming machine problem gamblers seek treatment

For example, 70-80% of those accessing Australian treatment services (Dowling et al 2005). But this is the figure for those who seek treatment, and is a self-selecting sample. How many problem gamblers of other types do not seek help?

e) Characteristics of gaming machines

Griffiths (1993) differentiated two types of factors in gambling:

- Situational characteristics - features of environment external to gambling: eg availability and accessibility of gambling activity, availability of cash withdrawal facilities;
- Structural characteristics - features inherent in gambling: eg speed of play, payout schedules.

Many features of gaming machines from these two categories reinforce the behaviour, as well as cognitive factors like the illusion of control.

Dowling et al (2005) felt that though gaming machines can be addictive, it is wrong to see them as the "crack-cocaine" of gambling.

A small amount of research has looked at the characteristics of gaming machines and their effect upon addiction.

i) Speed of play and payout

The average time on fruit machines between putting in the money and the result is 3-5 seconds. This gives a high event frequency. Then the individual can immediately play again leaving little time for reflection on losses. Pathological gamblers have been found to prefer faster games when the speed of machines has been experimentally manipulated (eg Loba et al 2001);

ii) Bet size

Gaming machines have inexpensive stakes. Problem

gamblers tend to want to bet more, and can lose interest if the bet size is too small (Dowling et al 2005). Furthermore, allowing machines to accept bank notes rather or as well as credit tokens purchased elsewhere encourages problem gamblers to spend more per sitting;

iii) Credited wins

Presenting details of credit won (and money spent) on the screen throughout the game was found, in the lab, to help pathological gamblers to stop playing. It had no effect on non-pathological gamblers (Loba et al 2001);

iv) Payout schedule

Frequent, small payouts generally is a powerful reinforcer (eg once every 3-6 trials);

v) Audiovisual effects

Pathological gamblers prefer more sound and visual effects in lab manipulations of machines (Loba et al 2001).

The question is how much do the characteristics of gaming machines initiate problem gambling and/or maintain it.

INTERNET ADDICTION

With the development of the Internet in recent years, research has turned to whether individuals can be addicted to its use. The possibility of Internet addiction was raised by Griffiths (1995). Soon afterwards, rating scales were developed to measure it: eg Pathological Internet Use (PIU) scale (Morahan-Martin and Schumacher 2000).

As time has passed, larger studies of Internet use and addiction have taken place, like Johansson and Gotestam (2004) in Norway. They interviewed 3237 12-18 year-olds by telephone or letter. The basis of the research was Young's Diagnostic Questionnaire (YDQ) (Young 1998) with eight items like "Do you feel preoccupied with the Internet (think about previous online activity or anticipate next online session)?" and "Do you feel restless, moody, depressed, or irritable when attempting to cut down or stop Internet use?". Answering yes to five or more questions is taken as addiction.

This study rated 1.98% as Internet addicted, and 8.68% as at risk (scoring 3-4 positive responses on the YDQ). The key symptoms of addiction were staying online longer than originally intended, using the Internet as a way of escaping problems, and being preoccupied with the Internet.

Also using the YDQ, Chou and Hsiao (2000) rated 5.9% of Taiwanese students as addicted, while figures for students in the US were nearer 10% (Morahan-Martin and Schumacher 2000).

Internet use is ever-increasing, and studies are very quickly dated. Cao and Su (2006) recorded an addiction rate of 2.4% among high school students (12-18 years old) in one city in China using the YDQ. On the Eysenck Personality Questionnaire (EPQ) adapted for China (Gong 1984), addicts had significantly higher P (psychotic scale) and N (neurotic-stable scale) scores than the rest of the sample. The addicts also showed problems in time management, and more potential psychiatric problems.

OTHER BEHAVIOURS

Two other behaviours are of particular interest to researchers: exercise (eg Allegre et al 2006), and shopping/buying. The latter seems to be associated with anxiety, depression, and obsessive-compulsivity (Rodriguez-Villarino et al 2006). Compulsive Buying Disorder, as it is called in DSM-IV, is estimated to occur in 1% of the US population, and predominantly in women (Bullock and Koran 2003).

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ISSUES

DRUG ADDICTION CAUSES MENTAL ILLNESS

There is the belief that substance abuse can cause mental illness, and, in particular, psychosis. Recently this has become encapsulated in the debate about regular cannabis use causing schizophrenia. Whether this is the case, the issue is as much about the appropriate methodology to study such a question. Generally two methods are used:

i) Cross-sectional studies - This method involves comparing groups at a particular point in time. Usually individuals who have psychosis are interviewed to discover their past cannabis use. The rate of use is then compared to a general population sample.

ii) Longitudinal studies - With this method, a group of individuals are followed over time to see how many who regularly used cannabis then developed psychosis. It is better able to establish a direction of causality than cross-sectional studies. But this type of study is usually with small numbers of people only.

Ernst (2002) argued that the problem in studying this area was in establishing the exact relationship between cannabis use and schizophrenia. It could occur in a number of ways:

- Cannabis use directly causes schizophrenia;
- Cannabis use precipitates schizophrenia in vulnerable individuals;
- Cannabis use is self-medication of pre-existing schizophrenic symptoms;
- Cannabis use makes symptoms of schizophrenia more pronounced and thus easily diagnosable;
- Cannabis use cause a psychotic condition which appears to be schizophrenia but it is not;
- Cannabis use produces short psychotic episodes which appear as one episode of schizophrenia.

For Ernst, the "problem is extremely complicated and unlikely to be answered on the basis of the existing, mostly epidemiological investigations" (p336).

The most recent review of the issue by Moore et al (2007) found a 1.5 times greater risk of mental illness among regular cannabis users, and twice the risk for daily users.

Hickman et al (2007) calculated that, with increasing cannabis use in England and Wales, by 2010, between 10 to 25% of new schizophrenia cases could be due

to cannabis.

SUBSTANCE ABUSE AND OLDER ADULTS

Almost automatically, substance abuse is perceived as a problem of the young. But alcohol and drug use "within older populations remains a significant issue, particularly in the context of the expected rise over the next twenty years of the population of older adults in the community" (Christensen et al 2006 p587).

Smoking

Generally individuals tend to stop smoking as they get older (Christensen et al 2006), but a sizeable number of older adults still smoke in some countries. For example, in a large survey of Chinese Singaporeans aged 45-74 years (Koh et al 2005), 22% of men and 3% of women had stopped smoking, but 36% and 6% respectively were still doing so. While in an Australian study of over 60s, only 6% were current smokers and 37% had given it up (Almeida and Pfaff 2005). In the USA, around 13% of adults over 60 currently smoke (Honda 2005).

Smokers have poorer physical health than past or nonsmokers (eg: 24% of British men over 60 who smoked had poor respiratory function compared to 6% of nonsmokers and to 10% of 18-39 year-old male smokers (Blaxter 1990). Smoking (and past smoking) is also associated with cognitive decline (Stewart et al 2006).

Historically, there has been a decline in smoking in Britain in all age groups. For men over 60 years, 44% smoked in 1974 and only 16% in 2001. For women of the same age, it is 26% and 17% respectively (Walker et al 2002).

Illegal Drugs

Arndt et al (2005) collected all the data for substance abuse programme admissions (both private and public) in the USA for 2001. They were 58073 admissions of adults over 55. This was about half the number aged 30-54 years. The number one reason for admission by older adults was alcohol (over three-quarters of cases), but much of the remainder was for heroin, cocaine and marijuana (21% of admissions).

Rockett et al (2006) screened patients in seven Tennessee hospital emergency departments in 1996-7: 20% of women and 7% of men older than 65 years used stimulants, and 15% and 12% respectively used opioids.

Because this study was based on toxicological analysis of saliva, urine or blood, it avoided the problem of undeclared substance use in self-reported questionnaire studies. The researchers interviewed participants before screening. For some substances, like alcohol, the screening results tallied with the self-reports, but, for example, with marijuana, 0.7% of men over 65 admitted its use in the last thirty days while it was found in 2.4% of screenings. For older women, the screening found nearly twice as much use of all substances compared to self-reports.

The older adults had the largest undeclared use of substance compared to other age groups. Some of this discrepancy will be due to memory problems or not knowing what was taken as well as lying.

Prescription Drugs

Addiction can occur with prescription drugs like tranquillizers (eg: benzodiazepines). Rockett et al (2006) found that a quarter of older adults used benzodiazepines. In an Italian study, around 80% of 65-84 year-olds surveyed had used them for more than a year (Balestrieri et al 2005).

Alcohol

The most common substance of abuse for all ages including older adults is alcohol. Studies show that, in absolute terms, less older adults abuse the substance compared to younger age groups. Table 9 gives some examples of recent studies.

Williamson et al (2003) used data from the GENESiS (Genetic and Environment Nature of Emotional States in Siblings) study of twenty-six GP practices in the UK to compare "binge drinking" among "singletons". Adults in their 50s were found to consume an average of 14.5 units (men) and 6.3 units (women) per week. This compared to 22.2 and 10.2 respectively for adults in their 20s.

Aira et al (2005) found that only 7% of men and no women over seventy-five years drank more than seven units per week.

The effect of alcohol use on cognitive decline tends to be that excessive drinking (> 21 units per week for men and > 14 for women) increases the risk of dementia (Christensen et al 2006). But this relationship is tempered by certain factors:

- Type of alcohol - light-to-moderate beer drinkers have a higher risk of dementia and light-to-moderate wine drinkers less risk compared to nondrinkers (Deng et al 2006);

<u>STUDY</u>	<u>RATES FOR OLDER ADULTS</u>	<u>RATES FOR OTHER AGE GROUPS</u>
Harford et al (2005) USA 2001 National Household Survey on Drug Use	DSM IV criteria: 0.8% men 0.5% women over 50 years	6.7% men/ 3.8% women (18-23 years) 3.8% men/ 1.2% women (24-29 years) 2.7% men/ 1.6% women (30-49 years)
Lukassen & Beaudet (2005) Canada Canada's Community Health Survey (CCHS)	"heavy drinkers" 5.3% over 65 years 9.3% men 1.4% women	37.5% (18-24 years) 22.8% (25-44 years) 15.7% (45-64 years)
Pirkola et al (2005) Finland Health 2000 Study	DSM IV criteria 0.6% over 65 years	5.0% (30-44 years) 3.5% (45-54 years) 4.0% (55-64 years)
Pirkola et al (2006) Finland Health 2000 Study	"active alcohol dependence" 2.1% over 65 years "remitted alcohol dependence" (not in previous year) 8.4%	active/remitted 49.8%/ 40.6% (30-44 years) 29.5%/ 31.3% (45-54 years) 18.6%/ 19.7% (55-64 years)
Rodgers et al (2005) Australia PATH Through Life Project	"hazardous drinking" (>28 for men/>14 for women "standard drinks" per week) men 6.8% women 5.7% (60-64 years)	men 6.6% (20-24 years) 6.4% (40-44 years) women 7.1% (20-24 years) 7.4% (40-44 years)

Table 9 - Examples of recent studies showing different rates of alcohol abuse between older adults and other age groups.

- Post-alcoholic abstinence - stopping alcohol

consumption can lead to cognitive improvements in months for men, but years for women (Dom et al 2005);

- Some benefits to light alcohol consumption - studies have found that light drinkers (1-6 drinks per week) benefit compared to nondrinkers (Christensen et al 2006).

For example, in an Australian study, Rodgers et al (2005) compared different levels of alcohol drinkers on a number of cognitive measures, including immediate recall on sixteen nouns, and recall of digits and repeat backwards. Light drinkers (< 14 for men and < 7 for women "standard drinks" per week) performed significantly better than nondrinkers (table 10), but performance declined with heavier drinking.

<u>COGNITIVE TASK</u>	<u>LIGHT DRINKERS</u>	<u>NONDRINKERS</u>
Immediate recall (out of 16)	7.89	7.35
Digits backwards	5.35	4.95

Table 10 - Mean scores on two cognitive measures between light and nondrinkers.

Polysubstance Use

In reality, many adults are abusing more than one substance at the same time: for example, alcohol and prescriptive medicine. In a Finnish study of home-dwelling over seventy-fives (Aira et al 2005), 44% admitted to drinking alcohol in the previous year, and nearly 90% of those used medication regularly, even those medications having potential interactions with alcohol, like painkillers. Men were the greater risk.

SUBSTANCE ABUSE AMONG ADULTS WITH LEARNING DISABILITIES

Sturmey et al (2003) estimated that up to 2% of adults with learning disabilities (AWLD) in the USA had a DSM defined "substance misuse disorder" as opposed to 8% of the general population. AWLD are less likely to use substances, but for those who do, the risk of abuse is high (McGillicuddy 2006).

Cigarette smoking, though, is similar in prevalence to the general population, if not higher, among adolescents and AWLD (particularly those living in institutions) (McGillicuddy 2006).

In terms of alcohol, studies have found that AWLD tend to consume less than the general population: for

example, in the US, 39% had had at least one drink in the past month compared to 50% of the general population (Substance Abuse and Mental Health Services Administration 2005 quoted in Barrett and Paschos 2006).

While in the UK, no AWLD drunk more than the Government's recommended weekly alcohol limit compared to 30% of men and 15% of women without learning disabilities. From a different point of view, 60% of men and 85% of women with learning disabilities are abstainers from alcohol (Robertson et al 2000).

Most of the research is questionnaire-based, and is limited by the respondent's understanding of the question, definitions of terms like "substance abuse" and "learning disability", the sample used, and being self-reported (or other-reported for adults with more severe learning disabilities). AWLD are a population with memory and cognitive deficiencies which can limit the recall of factual information (McGillicuddy 2006).

There is a common misconception that AWLD are at a greater risk of alcohol abuse. But Barrett and Paschos (2006) could find no evidence for biological predisposition among AWLD, and other factors, like susceptibility to peer pressure, and low self-esteem, are just as relevant to the whole population.

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